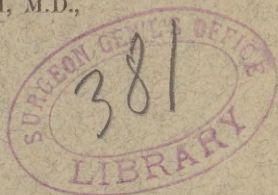


Dep
Putnam, (J. J.)

Lead in the Urine.

THE FREQUENCY WITH WHICH IT IS FOUND, AND
CERTAIN POINTS IN THE SYMPTOMATOLOGY
OF CHRONIC LEAD-POISONING.

By JAMES J. PUTNAM, M.D.,
OF BOSTON.



*Reprinted from the Boston Medical and Surgical Journal
of July 28 and August 4, 1887.*

BOSTON :
CUPPLES AND HURD, PUBLISHERS,
NO. 94 BOYLSTON STREET,
1887.

ON THE FREQUENCY WITH WHICH LEAD IS FOUND IN THE URINE, AND ON CERTAIN POINTS IN THE SYMPTOMATOLOGY OF CHRONIC LEAD POISONING.¹

BY JAMES J. PUTNAM, M.D.

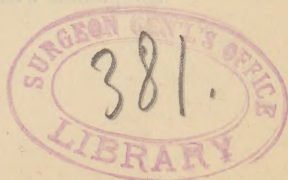
For a number of years past, several of my colleagues and myself have had the urine of patients with a variety of obscure diseases of the nervous system examined for lead, and have been surprised at the frequency with which it has been found to be present.

Wishing to learn whether this occurred only in certain classes of cases, or in the healthy and the sick alike, I have tried of late, with the valuable aid of Professor E. S. Wood, and of Dr. A. M. Comey, of Harvard University, to have these analyses made in a more or less routine fashion, though still giving prominence to the cases in which I had first found lead the most frequently.

The whole number of urine-analyses on which I have to report is eighty-six. In none of these cases were the classical symptoms of lead poisoning, such as colic, marked cachexia, characteristic cerebral attacks, characteristic paralysis, blue-line on the gums, unequivocally present. Twenty-six of the cases have been reported in two previous communications,² but are here re-analyzed. All of these patients took five or ten grains of potassic iodide three times daily for four or five days, and then collected a quart of urine, which was acidified with a little acetic acid and sent for examination.

¹ Read before the Association of Physicians and Pathologists, June, 1887.

² Boston Medical and Surgical Journal, 1883, CIX, No. 2. Journal of Nervous and Mental Disease, 1887, XIV.



tered, and re-precipitated in a perfectly clean test-tube with dilute sulphuric acid. The test-tube is allowed to stand twenty-four hours, and is then twirled gently between the fingers. The sulphate of lead, if present, rises in a delicate spiral.

Dr. Wood believes that this method is exceedingly delicate, and his opinion is borne out by the frequency with which lead has been found when not expected, and by the fact that he has rarely failed to find lead in cases of undoubted poisoning, which is by no means the experience of every investigator.

Such cases do, however, occasionally occur, under this as under all the other methods, and a negative analysis of the urine, especially a single analysis, is not a certain proof that the metal is really absent from the body.

Of the whole number of (eighty-six) cases, lead was found in the urine in forty-eight; in thirty-eight it was not found. If, for the sake of determining more accurately to what extent the presence of the lead is due to an exposure no greater than that to which the whole community is liable, we strike out those cases where the patients, through their profession, or some evident source of poisoning, were known to have been unusually exposed, we have left thirty-two cases in which lead was found, to be compared with thirty-eight where it was not found. We must conclude, so far as these figures are a guide, that, at the outside, in not more than fifty per cent. of persons living in the community at large, can lead be detected in the urine.

It may, of course, be argued, as indicated above, that the failure to find lead still more often probably shows only that the process is imperfect, but it is also to be borne in mind, as affecting this question, that although all persons are doubtless exposed to lead, some persons may eliminate the poison more rapidly and completely than others.

The obvious solution of this question would be furnished by chemical analysis of the organs of persons not presenting symptoms characteristic of lead-poisoning. So far as I know this has never been systematically done, but the following bit of evidence from Dr. Thomas Stevenson, of Guy's Hospital, is worth quoting.⁵

"It has been recently stated by M. Armand Gautier, that ordinary persons take lead to the extent of at least $\frac{1}{130}$ grains daily in their food. My own extensive experience leads me to the conclusion that it is exceptional to find a trace of lead in the body, except in cases of lead-poisoning, but that, on the other hand, it is equally exceptional not to find traces of copper in the human body after death."

Dr. W. B. Hills, assistant professor of chemistry at Harvard Medical College, has also told me that in making analyses of the human tissues he had often found copper but had never happened to find lead. He thinks, however, this may indicate only that lead is present in minute quantities, not that it is absent.

To turn now to the special groups of cases, the *first group* (those in which no symptoms were present which could not be fairly attributed to other causes than lead), contains eleven cases.

Of these eleven cases, three were of persons in perfect health; two were cases of sciatica; one a case of muscular atrophy not characteristic of lead, and affecting but one arm; one was a case of acute localized neuritis; three presented vague symptoms of no especial importance; one was a case of abdominal sarcoma. I will also mention that Dr. Comey examined four samples of his own urine, though without taking potassic iodide, and found no lead.

In two of these cases lead was found, in nine it

⁵ Guy's Hospital Reports, 1882, xxvi, p. 483.

was absent. This group is obviously too small to be of much statistical value, and the history of at least one of the cases where lead was found, which was that of a patient with abdominal tumor, but of excellent previous health, makes it more than probable that that others of like kind could be found on longer search.

The number of cases in this group might be somewhat increased if we should include in it those now set down in groups two and nine, of chronic functional nervous diseases, the neurasthenias and the epilepsies, which are those which could be with the least probability attributed to lead. If we do this, we raise the number of cases with no characteristic symptoms to twenty-eight, in ten of which lead was found. This gives us a provisional proportion of one to three as indicating the probable frequency of occurrence of lead in discoverable quantities without causing signs of disease.

If we add still further to this first group, the third group, characterized by symptoms of debility, such as might be due to many causes, but also by slight muscular tremor, recurrent paræsthesia of the hands, and other symptoms pointing possibly to slight organic disease, we get thirty-seven cases, in nineteen of which lead was found. In other words, we come back to the original proportion of fifty per cent. in favor of lead in healthy persons, which is, I think, undoubtedly too large.

In the *second group of cases*, a variety of neurasthenic and mild mental symptoms were present, without marked signs of nutritive disease, local or general. This group comprises nine cases, in five of which lead was found, while in four it was absent.

The cases are not sufficiently alike to form a coherent series, and I do not care to dwell upon them longer than is necessary to point out that the literature of lead-poisoning and the history of one of these

cases in particular, lends support to the view that purely nervous symptoms may be due to lead, and may exist for a long time without the supervention of signs of marked organic disease. Thus, Leudet, describing the cerebral symptoms which he had seen among a large number of painters, says :⁶

“Several painters, before they had shown any signs of cachexia, suffered from mental delusions, confusion and hebetude.”

Insanity of different types has been reported by a number of writers.⁷ Clevenger⁸ has described a case of melancholic depression with epileptiform convulsions in a lead worker, associated with blue-line on the gums “and other symptoms of lead-poisoning,” who recovered under potassic iodide and other treatment. Kiernan⁹ besides giving a number of references to other writers, gives an analysis of thirty cases of insanity apparently due to lead-poisoning, which were observed by himself. Some of these cases confirm the opinion which I have expressed. I will refer to only one case of those which I have observed.

It is that of an intelligent Scotch workman in a factory-town of Massachusetts, who had used for ten years water which was drawn through a two-inch lead pipe, from a well twenty-five feet distant, the end of the pipe remaining in the well. His family history was moderately neuropathic. His symptoms consisted in tremor of the hands, which had first been observed three years before, after the confinement and debility attending an operation for hæmorrhoids. Ever since that time he has felt weak and has worked

⁶ *Recherches sur les accidents causés par l’empoisonnement saturnine*. Rouen, 1882.

⁷ See Barteus ; *Allgem. Zeitschrift für Psychologie*, 37, 1881, Ulrich; ditto, 1883.

⁸ *Chicago Medical Journal and Examiner*, 1884; quoted in the *Journal of Nervous and Mental Disease*, April 1884, p. 308.

⁹ *Chicago Journal of Nervous and Mental Disease*, 1881.

but little, partly from that cause, but mainly because of distressing nervous symptoms, such as noises in the head, a feeling of soreness at the vertex, and, especially, frequent attacks consisting in a sort of aura sweeping over the body and head, the so-called "rush of blood."

His appearance was that of an excitable, but intelligent and naturally healthy man. The pulse and heart were normal. There was no anæmia, though this may have been masked by a natural fulness of the cutaneous vessels. The lips and tongue trembled slightly, as well as the hands. Lead was found in the drinking-water and twice in the urine. At a third examination it was absent. His improvement under treatment was marked and continuous.

This case leads us naturally to the *next group*, where it perhaps more properly belongs. This group of cases is allied to the last, but characterized by a greater prominence of disturbance of the organic functions. The general complaints were rather of lack of vigor, and of special nervous symptoms, rather than of general nervous instability or erythism, and yet there was no sufficiently marked organic disease to demand a special pathological classification.

In this group there are nine cases, in all of which, as it happened, lead was found in the urine. Five of them were examined for lead simply because they showed muscular tremor of the hands, or hands and lips, associated with some general debility or vague pains. Two, were cases of "recurrent paræsthesia" of the extremities, a symptom which it has of late been suggested may be due to slight multiple neuritis. If that is true, it might be supposed that a certain proportion of the cases would follow poisoning by lead, which imminently tends to attack the peripheral nerves. One of the patients in whom these symptoms of debility and tremor were well marked, and accom-

panied with increasing nervousness and sleeplessness, of four years duration, was a printer, and his urine contained a large amount of lead. His knee-jerk was very lively, especially on one side, but this symptom was less marked at a second examination, and there was no ankle-clonus. Its possible significance will be referred to later.

This group is related to that which follows (fourth group), in which I have put together the cases where symptoms of multiple neuritis were present, with others in which symptoms of chronic myelitis were associated with those of neuritis, and finally a number of cases characterized clinically as "spastic paraplegia," and due, perhaps, to degeneration limited to the motor tracts of the spinal cord. I have also included two cases apparently of multiple sclerosis of the spinal cord.

In this group of cases the chief interest of this investigation centres. For if it can be shown that lead poisoning leads to the symptoms of spastic paraplegia, or to some forms of it, especially those which are somewhat irregular in their clinical history, and are associated with signs of peripheral neuritis, on the one hand, and with more or less disorder of the general nutrition of the body, on the other, we shall have made a practical gain in our knowledge of a distressing disease.

This group of peripheral, mixed peripheral and spinal, cases contains nineteen cases, in all but four of which lead was found in the urine.

Of the four in which no lead was found, one was a case with chronic progressive spastic and ataxic symptoms, attended with difficulty in the retention of urine. The patient was a young girl, and the case probably belongs with the developmental diseases of the spinal cord. The other three were pretty typical

cases of spastic paraplegia, one of them associated with some muscular atrophy of the arms. The cases in which lead was found were characterized by such symptoms as the following:

(1) Paræsthesia of the hands and feet (two middle toes), twitching of the fingers, exaggeration of the knee-jerk, especially left.

(2) General feebleness; paræsthesia in legs with slight anæsthesia; twitching of muscles of legs; marked improvement under treatment; malarial history.

(3) Spastic paraplegia; cramps and twitching of the calves; knee-jerk and ankle-clonus exaggerated; history of chancre; lead found twice in urine and a considerable quantity also in drinking-water.

(4) Trembling of hands; sense of coldness and numbness in toes; lancinating pains in legs; fatigue on exertion.

(5) Marked progressive spastic paraplegia with myosis and loss of pupillary reactions; ataxia and some atrophy of hands.

(6) Progressive weakness and stiffness in legs with diffused and almost universal pains; marked tremor; great improvement for a time under treatment.

(7) Temporary pain in chest with slight dyspnœa; progressive numbness, heaviness and weakness in legs; eventually rather rapid recovery.

(8) Numbness in feet and legs with impairment of strength; tremor of hands and tongue; some wasting of small muscles of hands; temporary retention of urine.

One case is of peculiar interest as an instance, probably, of very severe and wide-spread neuritis of a type unusual in lead-poisoning, and characterized by a very marked ataxia; of motion, in short, a pseudo-ataxia of peripheral origin. It was reported briefly in the *Journal of Nervous and Mental Diseases*, January, 1887 (Case VIII.)

The next case is, I believe, of a similar though less serious character. It is that of a young lady¹⁰ of fifteen, of excellent previous health, who during the six months previous to my examination, which was in April, 1887, had shown successively the following symptoms: diffused headache of moderate severity; numbness and prickling of right hand; temporary difficulty in completely controlling the sphincter of the bladder; some months later, strabismus coming on gradually and slowly increasing, attended by dizziness, confusion of eye-sight and eventually by double vision; at the same period, numbness and prickling of left hand, especially at the ends of the fingers, continuing day and night, but eventually passing away; moderate ataxia of both upper and lower extremities; grasp of the left-hand weaker than that of the right, and small muscles of hand perhaps slightly atrophied; exaggerated knee-jerk, and trace of ankle-clonus; impairment of motions of both eyes, especially right, almost or wholly confined to muscles supplied by third pair; steady and rapid improvement, apparently aided by potassic iodide; eventually complete recovery.

That these are instances of multiple neuritis, not as is the rule with lead-poisoning confined to the motor tracts, but involving the sensory as well, and thus causing the ataxia, cannot, I think, be doubted. Analogous if not exactly similar cases have been reported under the name of pseudo-tabes, due to peripheral neuritis from alcohol and arsenic¹¹ and Renaut,¹² in his excellent monograph on chronic lead-poisoning, reports one of the same kind, evidently due to lead, which was observed by Raymond, who says in another place, that he has seen three cases of the kind.

¹⁰ Seen in consultation with Dr. P. Wadsworth, of Malden, Mass.

¹¹ *Gazette Medicale de Paris*, 1876, Vol. 35.

¹² On Pseudo-tabes from Arsenical Poisoning, etc. Charles L. Dana. *Brain*, Part xxxvi, January, 1887.

The next case is likewise interesting as showing symptoms of progressive spinal character, involving almost exclusively the motor functions and associated with marked anaemia. It resembled in these two respects another case which I have placed in a different group, characterized by progressive anaemia, progressive transverse myelitis and death.

The patient had been a carriage-painter for thirteen years. His present symptoms consisted in "numbness" of the feet and legs throughout their full extent; fatigue in walking and marked muscular weakness, great constipation; similar symptoms in arms but to a less degree; no pain; marked exaggeration of knee-jerk and ankle-clonus. The duration of the symptoms was about one year and a half. No albumen was found in the urine, nor other signs of chronic nephritis. The skin was very pale, with a yellowish cast. There is no distinct ataxia, and yet the movements are rather exaggerated; no history of colic; no apparent weakness of the extensors of the fingers; no blue-line. Lead was found twice in the urine. The patient improved markedly under iodide of potash and a large quantity of food, but lately has failed steadily. The exaggeration of the knee-jerk, and the presence of ankle-clonus, in this last and some of the preceding cases leads us to (group IV) the cases of spastic paraplegia, amyotrophic spastic paraplegia, and paraplegia with ataxia, to which I have already alluded. These, together with the cases of probable multiple spinal sclerosis, number ten, and in all but three of them lead was found in the urine.

That some of the characteristic symptoms of spastic paraplegia are occasionally due to lead-poisoning can, I think, be established beyond much doubt, though the fact is not generally recognized, all the writers on the subject, from Tanquerel down, speaking of the

paralysis of the legs as characterized by atrophy and muscular relaxation, like that of the upper extremity, not by rigidity and exaggerated deep reflexes. The former condition is certainly the rule, and for the simple reason that the paralysis of the legs, like that of the arms, is usually due to peripheral neuritis. Brissaud, for example, takes this view, in his recent and excellent monograph on the toxic paralyses,¹³ yet, while calling attention to the fact that the so-called deep reflexes are habitually diminished or lost, he says that in exceptional cases they are exaggerated, owing as he thinks, to a sort of associated condition of spinal irritation.

Brissaud also points out that the interesting observations on the effects of poisoning by the different species of lathyrus, in the southern countries of Europe, and especially the investigations by Bouchard and Proust, show that the typical symptoms of lathyrism consist in spastic paraplegia, with exaggerated deep reflexes and without marked sensory symptoms. Ergot¹⁴ also causes pronounced spastic paraplegia as its typical sign of its poisonous action.

Besides this spastic form, there is a flaccid form of lathyrism paralysis which Brissaud thinks is perhaps of peripheral origin, and may justify the ranking of this poison under the same head with lead, arsenic and alcohol. The question is whether these latter agents also, or at any rate lead, besides the flaccid paralyses or peripheral origin may not sometimes cause a typical spastic paraplegia.

I am well aware that the presence of ankle-clonus and exaggerated knee-jerk cannot be taken as proof of the existence of spinal sclerosis. They are found exaggerated in hysteria and neurasthenia, acute and

¹³ Des Paralysies Toxiques. Paris, 1886.

¹⁴ Tuzek, Arch. Für Psych. und Nervenkrankheiten, 1887, xviii, 329.

chronic, and I have seen both quite recently in two cases of severe anamia, one following rheumatism and one of unknown origin. They may, in short, indicate only a functional, nervous hyperexcitability. Such may have been the origin of the symptom in a case reported by Dr. Dercum, of Philadelphia,¹⁵ where a very large amount of lead was found in the urine, and which was characterized otherwise by the following symptoms; loss of strength and weight for two years; great nervousness; insomnia; severe itching of the skin; numbness of the right arm; indigestion; cramps in the abdomen; exaggeration of the knee-jerk.

Finally, Möbius, and Strümpell have reported two cases supposed to be instances of multiple neuritis, in which this symptom was present, and perhaps another example of this kind is furnished by the following case, which was reported by Dr. S. G. Webber¹⁶ as probably an instance of lateral sclerosis due to lead, but which seems best interpreted as peripheral polyneuritis with exaggerated knee-jerk.

“Female patient, married, twenty-three years old; numbness and pain below the elbows and knees, for three months past; swelling of legs; at time of examination, paralysis of feet in extension, with pain on passive motion; delayed sensation from feet; complete paralysis of extensors of wrist and fingers, and incomplete paralysis of the rest of the muscles of the arm below the elbow; diminution or loss of Faradic reaction; urine contains considerable lead; knee-jerk very greatly exaggerated, improvement under treatment.”

Setting aside for the moment the question whether we are justified in assuming that the pronounced exaggeration of the knee-jerk and the presence of the ankle-clonus in these cases is really due only to a

¹⁵ Philadelphia Medical News, January, 1887.

¹⁶ Archives of Medicine, New York, Vol. viii, 1882, No. 1.

general nervous erythism, or to an irritability of the spinal cord, let us glance at the remaining evidence that this symptom and other signs of spastic paraplegia are seen in typical lead-poisoning and in the cases where lead was found in the urine.

The first case is that of T. C., a stone-cutter, from Quincy, who came to the Massachusetts General Hospital on May 5, 1887. He reported that he had a chancre four or five years ago, followed he thinks, by secondary symptoms, and used also to drink a good deal of liquor, but not for three or four years. Except for this his previous health had been good, and he had not at all the appearance of a person suffering from the cachexia of alcohol. For three or four years he has not felt well, and has been subject to "jumping" pains in the head, back and legs, especially about the knee; also pains in the abdomen, not always attended with indigestion.

Three months ago he began to lose power in the extensors of the fingers and thumb of the left hand. This increased steadily but is still incomplete, the little finger being extended fairly well; the others in the order in which they follow. The extensores carpi radialis and ulnaris are both deficient, the ulnar extensor, as is usual in lead paralysis, acting the best. There is slight tremor on attempted extension of the fingers. The muscles of the right arm are in a similar but much less serious condition; that is, there is a slight tremor and incomplete extension of the fingers. The electric examination shows loss of Faradic reaction in the extensor communis digiti of the left arm, and R. D. at three M. A. The Faradic reaction of the flexors of the left arm, and of both flexors and extensors of right arm is normal. The knee-jerk is exaggerated on both sides, though more left than right, and there is a trace of ankle-

clonus, increased by "reinforcement." There is no marked spastic gait, but at times his legs feel stiff.

The second case is as follows: Male patient; works at grinding up old articles of manufactured rubber (in the making of which litharge is used); has had severe abdominal pains for the past four months; constipation; tenderness on pressure over the abdomen; fatigue in walking; insomnia, due he thinks to the pains; yellowish cast to conjunctiva and complexion; *marked blue-line* on the gums; exaggeration of the knee-jerk, and trace of ankle-clonus.

I am indebted to Dr. R. T. Edes for the notes of the following cases:

(1) Jennie C., aged twenty-two. Sharp pains in legs, with numbness and inability to walk; creeping sensations in the hands; muscular tremors; exaggerated deep reflexes; lead in urine.

(2) John D., aged twenty-six. Pains in thighs, and numbness of legs; staggers a little in walking and standing with eyes closed; patella reflex exaggerated; ankle-clonus. No syphilis. In this case the urine was not examined for lead, but it is some evidence in favor of its presence that the patient worked in rubber, an industry in which lead is largely used, and which has furnished me with a typical case of spastic paraplegia, and two cases of typical lead-poisoning.

Finally, we come to the instances of typical spastic paraplegia and allied cases, where lead was found in the urine.

Of these the first is the case of a plumber, who besides a typical spastic paraplegia, which was of a year and a half's standing, had considerable impairment of hearing and eyesight, of the same duration, slight but distinct trembling or twitching of the lips and muscles of the face, in talking, and looked pale and rather sickly.

In the second and third cases the symptoms of spastic paraplegia were also well marked. A certain degree of support to the motion of causation by lead, besides the finding of lead in the urine, was furnished by the fact of considerable improvement, partly under treatment, partly without treatment, so that one of the patients was eventually able to resume work though not cured.

The significance of this improvement as a sign of poisoning rather than of progressive degenerative disease could easily be overrated. Such cases do sometimes improve, at any rate, for a time, and in one of these the improvement seemed more or less independent of the taking of potassic iodide. Still, improvement for any considerable length of time is not common in the run of cases of chronic myelitis of any form. It is also worthy of note that, as Tuczek¹⁷ has pointed out, the spinal sclerosis due to ergot, which gives rise to these same symptoms of spastic paraplegia, shows a rather remarkable tendency to remain without progressing, though without much improvement, when the patients are withdrawn from the influence of the poison.

In a fourth case the patient was a painter, and besides his spastic gait and exaggeration of the deep reflexes, he suffered from attacks of dizziness and increased frequency of micturition. A small trace of lead was found in the urine. The symptoms seemed to indicate an extension of the disease beyond the limits of the spinal cord.

In a fifth case the spastic symptoms were accompanied by signs pointing to multiple sclerosis; ataxia of arms and legs; almost universal paræsthesia and anæsthesia, especially in patches; great impairment of muscular sense; excessive hyperæsthesia to

¹⁷ Arch. fur Psych. und Nervenkrankheiten, 1887, xviii, 329.

contact with hot or cold objects; transient hemiparetic attack; tremor on effort. These was exaggerated knee-jerk but no ankle clonus; no cachexia suggesting lead.

On the other hand, in three typical cases of spastic paraplegia, one of them complicated with atrophy of the muscles of the hands and arms without contracture, no lead was found. The test for lead was also applied, and none found in another case, which might be classed under the head of multiple sclerosis, but this was evidently not a case of sclerotic inflammation of the ordinary type, but rather of some hereditary degenerative process, the patient being a young girl and the case peculiar in several respects.

I am by no means prepared to assert that a clear case is made out for the causation by lead of typical chronic spastic paraplegia, or of ataxic paraplegia. I would only claim that the cases I have reported indicate that as a direction in which to look in the future. The fact that ergot and lathyrus, cause the same group of symptoms, and that syphilis leads to another form of spinal sclerosis, rather encourage us to look favorably upon the evidence adduced, and what we already know of the pathology of lead-poisoning does not make such a connection intrinsically improbable.

It is perfectly true that the great weight of pathological evidence is in favor of the view that the typical lesion of lead paralysis is an inflammation not of the spinal cord, but of the peripheral nerves, but it is equally true that the nutrition of the spinal cord is sometimes more or less grossly impaired (in six out of fourteen cases, as Dr. Birdsall¹⁸ made out from an analysis of autopsies, published up to 1881. See

¹⁸ Journal of Neurology and Psychiatry, New York, 1881.

also Schultze,¹⁹ Oppenheim,²⁰ Brissaud,²¹ and Robinson.²²)

Again, although it is true that typical *outspoken* lesion in lead-poisoning is usually confined to the peripheral nerves, and even to the peripheral extremities of the peripheral nerves, yet, as is pointed out by Oswald Vierordt, the writer who has spoken the latest word on the subject,²³ lead paralysis is, after all, a "system" disease, not a general neuritis, and as such probably affects in some degree all the segments of the motor system of which those special nerves form a part. The injury to the spinal cord probably effects no more, in most cases, than an impairment of its normal nutritive influence upon the peripheral nervous and muscular apparatus. If this opinion is correct, the involvement of the spinal cord may be a matter only of more or less, and it is not inconceivable that the antero-lateral pyramid tracts of the spinal cord, though not forming a part of the lower nerve segment (Gowers) which is primarily involved in lead-poisoning, might be secondarily involved, as so often happens in progressive muscular atrophy; or might be primarily and separately attacked; or, finally, the disease in them might be due to thickening of the vessels so common in lead-poisoning. Such reasoning is, however, of secondary importance in the absence of more definite clinical and pathological evidence.

In contrast with these cases where the motor spinal tracts were involved and where lead was so often present, I would mention that in three cases of local ataxia; five cases of progressive muscular atrophy; and one which was a case of either acute symmetrical

¹⁹ Ueber Bleilähmung. Arch. für Psych. und Nervenkr, xvi, 1885. p. 791.

²⁰ Arch. für Tsyeh. und Nervenkrankheiten, xvi, 1885, page 476.

²¹ Loc. cit.

²² Brain, Vol. vii.

²³ Arch. für Psych. und Nervenkr. xviii, I, 1887.

neuritis, or of polymyelitis of the adult, no lead was found.

The next group of cases examined for lead is characterized by more or less distinctly localized cerebral or cerebro-spinal symptoms. This group contains seven cases, in all of which lead was found. Three of these cases may be excluded because the disease was fairly assignable to other causes. The other cases are not sufficiently alike to make it worth while to consider them together, and I will therefore postpone the enumeration of them to a future time.

The next group is that of the epileptic cases. This contains eight cases, in only three of which lead was found. Of these three patients, one was a young lady in good circumstances, who was subject to peculiar attacks of petit mal, consisting in a sensation of nausea with little or no impairment of consciousness, and occasional attacks of temporary blindness of one eye. She was about twenty-five years of age, and had had the attacks for a number of years; no sign of lead cachexia was present, but lead was found twice in the urine, once in considerable quantity. At a third examination it was not found.

Of the second case I have no full notes. The patient was sixteen years old and had attacks of petit mal about once a week for three years: whether cachexia was present or not I do not know.

The third case seems to me of special interest. It is that of a student about twenty-six years of age, who had suffered during the past year from numerous attacks of petit mal, and one severe attack of outspoken epilepsy. There was no family predisposition to be discovered, and the patient could not remember ever to have had such seizures before, except that once, when about fifteen years old, while sitting at his desk reading or writing, his vision suddenly left him for a moment,

as he thinks. The patient is decidedly pale, though this is in part natural; he looks rather feeble; there is slight tremor of the hands; he also did very well under treatment with potassic iodide, and bromide. The urine was examined three times for lead and twice it was found. I was inclined to make light of the connection between the lead and the epileptic symptoms until I found to my surprise, on asking the patient to extend the fingers and wrist to their full extent, to find that the three smaller fingers of both hands drooped considerably, those of the right hand more than those of the left, and that, on the electric examination of the muscles, the *extensores communes digitorum* of the right arm responded less easily to both galvanism and faradism than the corresponding muscles of the left arm. The interest of these cases centres on the inquiry which they suggest, whether lead can cause epileptic symptoms for any considerable length of time without signs of marked cachexia, or other serious phenomena of lead-poisoning. This condition, though certainly rare, seems to be occasionally met with.²⁴

The epileptic attacks recorded as due to lead are usually severe and prostrating, but of course lighter attacks really due to lead may have been referred to some other cause. It seems to be certain that the children of parents with lead-poisoning suffer from convulsions, in which as a rule they die in infancy. That epileptic attacks of the type of *petit mal* may, however, be present for a long time without any serious cerebral symptom is well illustrated by the following case which I have recently observed.

It is that of a young woman twenty-seven years of age, employed in a shoe-shop in a country town, and

²⁴ See Leudet; *loc. cit.*; also Schultze, Dissertation, Breslau, 1885, p. 21; Berger, *Berl., Kl. Wochenschr.*, 16, March, 1884.

drinking water which came, as is so common in country towns, through twenty feet of lead-pipe from a sunk well, the end of the pipe lying in the well. Her symptoms date back about three years. At this time her health began to fail, but she showed no more definite symptoms of lead-poisoning than frequent pains in various parts of the body, and in the abdomen, the latter, to be sure, so severe that she was thought by some of the physicians whom she saw, to have inflammation of the bowels. For two or three years she had, at short intervals, what she called "fainting spells," characterized by a loss of consciousness for two or three minutes, preceded by a sense of dizziness, but never attended by convulsions, and leaving her, after a few moments, in her normal condition. She also suffered a good deal with diffused headaches, and dizziness. After this state of things had continued for about two years, sometimes better and sometimes worse, she had a violent cerebral attack, with prolonged loss of consciousness, convulsions and amaurosis, lasting for nearly a week, and this was followed, after a time, by another similar, but less severe attack, and later by characteristic and severe palsy of lead, from which she recovered, only to be attacked by a similar paralysis of other muscles than those first affected. At the time of my examination there was an indication of blue line on the gums, incomplete extension of the fingers and wrist, and paralysis of the small muscles of the hands, with atrophy and reaction of degeneration.

Besides these groups of cases which I have reported, there are a number which do not fall under any single heading, with the recital of which I will not take the time of the association.

Looking back over all the groups, it may be pointed out: *first*, that the urines of persons known to be in

perfect health, though their number is, to be sure, but few, were almost all free from lead; *second*, that in the cases of functional nervous disease, neuræsthenia and epilepsy, the cases in which lead was found were in a decided minority; *third*, that in proportion as these nervous symptoms were associated with signs of organic disease, the proportion of cases in which lead was found became greater; *fourth*, that among the cases of organic disease, those in which lead was found with the greatest regularity were cases presenting symptoms of chronic diffused neuritis and chronic myelitis, especially of the motor tracts of the spinal cord; *fifth*, that among these cases, those which presented the least sign of cachexia, were those in which lead was not present; *sixth*, that the cases of progressive muscular atrophy showed no lead; *seventh*, it should be remembered that these conclusions are provisional only, and may be invalidated if it should turn out that lead is more frequently found in normal urine than has been hitherto shown to be the case. Finally, I would suggest, as worthy of future study, the question whether lead may not cause epileptic symptoms of long continuance, without indicating its presence by other unequivocal signs.

In conclusion, I would call attention, as I have already done in a previous paper, to one occupation which has furnished me with several marked instances of lead-poisoning, and which, to my surprise, I cannot find set down among the dangerous employments from this point of view, and that is the manufacture of articles of rubber.²⁵

²⁵ Lead has been found in the rubber ends of feeding-bottles, in Europe.

